

concentration.<sup>1</sup> Dahlquist *et al* were able to precipitate an episode of D-lactic acidosis in a patient with intestinal bypass surgery, by using a 25.08 MJ (6000 kcal) load containing 54% carbohydrate.<sup>5</sup> Similarly, Rosenthal and Pesce described a recurrence of abnormal neurological signs, together with high D-lactate concentrations, in a patient in whom enteral feeds were substantially increased.<sup>6</sup>

Our studies show the importance of the nature of the dietary carbohydrate substrate in the production of D-lactic acidosis. Manipulation of dietary carbohydrate with strict control of monosaccharides and oligosaccharides was successful in our patient and preferable to repeated courses of broad spectrum antibiotics, which may alter the colonic flora in such a way

as to impair valuable colonic salvage of non-absorbed nutrients.

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## Atopic eczema, hyponatraemia, and hypoalbuminaemia

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### Abstract

**We describe an infant with atopic eczema, treated with homoeopathic medicines, who presented with erythema and limb oedema. Concentrations of urinary and plasma sodium and plasma albumin were low. On conventional treatment he made a satisfactory recovery.**

Atopic eczema affects 5–10% of children under the age of 5 years.<sup>1</sup> We report an infant with atopic eczema, treated inappropriately with homoeopathic medicines, who became seriously ill with deterioration of the eczema and associated metabolic complications.

### Case report

A 6 month old boy presented to casualty with a one week history of generalised erythematous weeping skin and pronounced oedema of the limbs. His parents had refused hospital admission when seen four days previously. Since 1 month of age his skin had been dry and eczematous. Homoeopathic medicines were prescribed for his eczema by a registered homoeopath. The eldest of his three brothers who had mild eczema in infancy had received similar treatment. Conventional treatment was repeatedly declined by the family, apart from a one week hospital admission at 4 months of age with bronchiolitis and eczema. He had received nine different homoeopathic medicines before admission including a six centesimal ( $10^{-12}$ ) dilution of trace metals (iron and arsenic).

Biochemical investigations showed low plasma and urinary sodium concentrations of

121 mmol/l and 10 mmol/l respectively and a low plasma albumin of 11 g/l (no proteinuria). Activities of liver transaminases and alkaline phosphatase and concentrations of plasma urea and potassium were normal.

Treatment was given with 0.45% sodium chloride solution, repeated albumin infusions, and intravenous flucloxacillin and benzylpenicillin. Intensive local skin care was started with potassium permanganate baths twice daily. Initially an ointment containing 1% hydrocortisone and 3% clioquinol (Vioform-Hydrocortisone; Ciba) was applied and then changed to one containing 0.0125% flurandrenolone and 3% clioquinol (Haelan C; Dista) applied twice daily to affected areas. A cream containing 1% hydrocortisone and 2% miconazole nitrate (Daktacort; Janssen) was applied three times daily to the perineum, and Diprobace cream moisturiser (Kirby-Warrick) was used on all areas of the body every two hours. Skin swabs initially grew *Staphylococcus aureus* and  $\beta$  haemolytic *Streptococcus Lancefield* group A. Repeat swabs also grew *klebsiella* and *Escherichia coli* and thus the antibiotics were changed to ceftazidime and gentamicin.

The weeping, erythema, and oedema of his skin gradually subsided and progress was satisfactory, apart from an episode of rotavirus gastroenteritis. He was discharged after three weeks with normal plasma sodium and albumin concentrations. On review at 10 months of age, he is thriving with mild eczema, which is now maintained on daily baths using an additive containing 5% acetylated wool alcohols and 63.7% liquid paraffin (Oilatum Emollient; Stiefel), 1% hydrocortisone ointment, and Diprobace cream.

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### Discussion

Acute exacerbation of atopic eczema with weeping red skin is usually caused by secondary infection and requires prompt treatment with appropriate systemic antibiotics. Hyponatraemia and hypoalbuminaemia are rare complications of atopic eczema and occur if weeping (exudation of serum) is chronic and persistent.

Recently the number of parents who seek alternative forms of treatment has increased with the growing popularity of 'natural' products. While homeopathic medicine has relieved some diseases,<sup>2</sup> in our opinion there is

little evidence to suggest it is helpful in atopic eczema. Thus caution is needed in the use of homeopathic medicines as the sole treatment for this condition. Severe exacerbations should be recognised and treated early by conventional medicines to avoid the risk of potentially life threatening complications.

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## Growth failure secondary to moyamoya syndrome

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### Abstract

**We describe a boy who presented at the age of 7 years with short stature due to hypopituitarism. Six months after starting appropriate hormone replacement treatment at the age of 8 he suffered his first generalised convulsion. Further neuroradiological investigation led to the diagnosis of moyamoya syndrome.**

Moyamoya is the arteriographic appearance of a filmy network of small vessels at the termination of the internal carotid artery. We describe a boy with hypopituitarism associated with moyamoya.

### Case report

The boy presented initially to our hospital at 18 months of age with obesity and developmental delay. He was the fifth child of unrelated, white parents (maternal height 0 SD, paternal height -0.9 SD) and was born at term by spontaneous vaginal delivery. The birth weight was 3450 g and there were no perinatal problems. The developmental delay was thought to be due to lack of parental stimulation and a physiotherapy programme was devised with support for the family. At 3 years of age he was admitted to hospital for investigation of persistent lower limb weakness but biochemical investigation excluded a myopathy. The family subsequently failed to attend hospital appointments.

At 7 years he was referred to the endocrine clinic with short stature, small genitalia, and headaches. On examination he was moderately obese with weight on the 25th percentile and height well below the 3rd percentile (-3.1 SD). There were no dysmorphic features. Blood pressure was 95/60 mm Hg. Fundoscopy and visual fields to confrontation were normal. He was alert and cooperative but clumsy with tendon reflexes preserved and flexor plantars. His penis was small and the testes were less than 1 ml volume.

Investigation showed a TW2 bone age of 3.4 years at chronological age 7.1 years. Anterior

pituitary function was assessed after intravenous injection of thyrotrophin releasing hormone (200 µg) and luteinising hormone releasing hormone (100 µg) with oral clonidine 150 µg/m<sup>2</sup>. Fasting venous samples were taken at 0, 20, 75, 90, 120, and 150 minutes. Plasma cortisol concentrations were 456, 440, 155, 215, 347, and 469 nmol/l respectively. Peak plasma growth hormone concentration was 1.6 mIU/l at 150 minutes and the peak plasma luteinising hormone and follicle stimulating hormone concentrations were 1.3 IU/l. Plasma thyroxine was 44 nmol/l with an abnormal, sustained rise in thyroid stimulating hormone (2.0, 13.0, 20.0 mIU/l at 0, 20, and 75 minutes) indicative of hypothalamic pathology. The basal prolactin concentration was raised at 642 mIU/l. These results confirmed hypopituitarism. Posterior pituitary function was not affected.

Treatment with growth hormone and thyroxine was started at the age of 7.4 years resulting in a rise in growth velocity from 2.3 cm year in the four years before treatment to 9.8 cm in one year on treatment. At 7.9 years he suffered his first generalised seizure and an electroencephalogram confirmed epileptiform activity. A radiograph of the skull showed amputation of the dorsum sellae; computed tomography showed a shallow pituitary fossa and suprasellar calcifications with obliteration of the cisternae but no well defined soft tissue mass. The third ventricle was deviated slightly to the left side. The radiological findings were not considered to be sufficient for diagnosis but the possibility of a craniopharyngioma was discussed. The seizures were controlled with carbamazepine and a magnetic resonance scan requested to allow better definition of the suspected tumour. Subsequently the child developed worsening headaches and at 8.4 years of age was admitted to hospital with blurred vision, ataxia, and increased seizure frequency. A repeat computed tomogram confirmed suprasellar calcification with highly attenuating tumour like tissue in the suprasellar region, now also extending to the ambient cisterns with

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